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THE PROTOZOAN PARASITES OF DOMESTICATED ANIMALS.

BY

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THE PROTOZOAN PARASITES OF DOMESTICATED ANIMALS.

By HOWARD CRAWLEY,
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INTRODUCTORY.

Attention was first forcibly called to the Protozoa as causes of disease by Pasteur, who showed that pebrine, the silkworm disease which broke out in France in 1845, was caused by a protozoan parasite. This disease is estimated to have caused a loss to the silkworm industry of France of $200,000,000 up to the year 1867. The silkworms of France, and of other parts of Europe as well, were practically all destroyed, and the industry was only put on its feet again by the importing of uninfected eggs from Japan.

The second great discovery in the science of protozoology was made in 1882 by Laveran, then a French army physician. This was that the cause of malaria in man is a protozoan which invades and destroys the red blood cells, in this way causing the anemia which is one of the symptoms of that disease. The later discovery that malaria is carried from man to man by a mosquito, and in no other way, has enabled medical science to indicate the method whereby it may be eradicated. The association of malaria and swamps has no other signification than that the swamps furnish breeding grounds for mosquitoes.

The third great discovery was that of Theobald Smith, who, working at Washington in 1892, found the cause of Texas fever of cattle to be a blood parasite allied to but not identical with the malaria parasite of man. This parasite also attacks and destroys the red blood cells, causing an anemia so profound that the blood count may fall to one-third or less of the normal. The parasite is conveyed from one animal to another by the cattle tick, and Texas fever can not exist in a region where there are no cattle ticks. Unfortunately for the agricultural interests of the United States, the tick is present throughout the Southern States, and as a consequence almost all southern cattle harbor the parasite in their blood. Since, however, they become inoculated as young calves, when possessing a high degree of resistance to the parasite, they become naturally vaccinated and seldom suffer from the disease in an acute form. On

the other hand, northern cattle, which are not naturally vaccinated, are highly susceptible, readily acquire the disease, and usually die if they become infected. It follows from this that it is impossible to move cattle with safety from the North to the South or vice versa without taking certain troublesome precautions. This, in itself, is a serious loss to agriculture, and moreover southern cattle, both on account of the constant presence of the parasite in their blood and the presence of sometimes large numbers of ticks on the skin, are thereby debilitated. The cattle tick is estimated to cost the live-stock industry of the United States from $30,000,000 to $50,000,000 a year.

In Africa a disease of man, appropriately termed sleeping sickness, has been known to civilized man for over a century. Originally confined to the West Coast, it has recently spread eastward and is now a serious menace to the development of Central Africa. In 1902 the cause was discovered to be a trypanosome, a flagellated blood parasite belonging to quite a different group from those before mentioned. This trypanosome is carried from man to man by a biting fly. While cases of sleeping sickness have been cured, the disease is ordinarily fatal, and yearly causes thousands of deaths among the negroes of Africa.

The instances above mentioned show very clearly the importance of the Protozoa from the economic standpoint, both as regards disease in man himself and in animals upon which he depends. The group, long neglected, is now being studied by a large number of investigators, particularly in Europe and Africa. In the United States not so much attention has been paid to the Protozoa as pathogenic organisms, doubtless owing to the fact that this country has so far escaped the ravages of the great trypanosomiases. Further, Texas fever, probably the most serious protozoan disease attacking domesticated animals in the United States, is rather easily controlled, owing to the very limited powers of dispersal possessed by the carrier, the cattle tick. Nevertheless, it is impossible to lay too much stress on the importance of the study of the Protozoa as causes of disease.

In the first place, Protozoa probably do a very considerable amount of harm to the live-stock industries in the United States which altogether escapes attention. For instance, a number of protozoal diseases are always enzootic, and hence do not attract as much attention as would be the case with an epizootic. But the sum total of loss caused by a large number of small outbreaks might easily be considerable. In the second place, the immunity enjoyed by the United States to the great animal trypanosomiases of Africa, Asia, and South America is probably more a matter of good fortune than anything else. The impression that these diseases can exist only in tropical countries is wholly erroneous. Their existence and spread depend exclusively upon the presence of the proper carrier, which appears in all cases to

be a fly, and in the warm season flies are just as abundant in this country as they are in Asia, Africa, or South America.

It so happens that the tsetse flies, which act as carriers for the African trypanosomiases, are not present in the United States, and accordingly it is generally supposed that there is no danger of these diseases spreading here, even were they to be introduced. It is by no means certain, however, that the African diseases are absolutely dependent on the tsetse flies, and those of Asia and South America certainly are not, since tsetse flies are not present on either of these continents. It may be laid down as a rule that the presence of any biting fly constitutes a source of danger in the presence of a trypanosomiasis, and, as is well known, biting flies are very abundant in America, common examples being horse flies, the stable fly, and the horn fly.

Therefore the Protozoa are of great interest to the live-stock industry of the United States, and it is the purpose of this article to give a brief account of those which are parasitic, with particular reference to those parasitizing domesticated animals.

DESCRIPTION OF THE PROTOZOA.

As their name indicates, Protozoa are primitive animals; they are, in fact, the simplest known forms of animal life. Like the corresponding forms of plant life, the Protophyta, they are unicellular, and in this respect these two forms differ from all other animals, which are multicellular. The Protozoa, though generally larger than bacteria, are almost all so small that they can be seen only with the aid of the microscope. They are widely distributed in nature, being found almost everywhere, and, like bacteria, some species are free living while others live as parasites upon or within other animals.

A protozoan consists of a mass of protoplasm containing a nucleus. Although so simple in structure, they exhibit practically all of the biological phenomena characteristic of the physically complex higher animals. Protozoa take in food, digest and assimilate it, excrete waste products, grow, and reproduce.

Reproduction is effected in two ways, as follows: (1) By bipartition, or division into two; (2) by endogenous multiplication, or division into many. In the first, the nucleus first divides into two parts, the protoplasm follows suit, so that each daughter cell is a replica of the mother cell, and indeed consists of one-half of it. Division of this sort occurs when the protozoan has reached its maximum size, and is shown by trypanosomes.

In endogenous multiplication the nucleus divides into a large number of small bodies. Each of these then collects around it a small part of the protoplasm, and the mass thus constituted breaks up into a number of small bodies. These are often spoken of as spores. This is the usual mode of division in the Sporozoa.

Under certain conditions Protozoa conjugate, conjugation consisting in the fusion of two individuals. These are termed the gametes, and when two gametes conjugate they form a zygote. In certain Protozoa, the two gametes are to all appearances exactly alike, whence the process of conjugation is termed isogamy, or the union of like gametes. In others, the gametes differ, whence the process is called heterogamy, or the union of unlike gametes. In many cases of heterogamy the gametes are of very different sizes, in which case the larger is known as the macrogamete and the smaller as the microgamete. Macrogamete and microgamete correspond, respectively, to the egg and spermatozoan of higher animals.

As the result of any one of several influences Protozoa encyst. The active, usually motile, organism becomes inert, assumes a spherical shape, and secretes a resistant sheath, often shell-like. In this condition the protozoan is resistant to drought and can also resist the action of substances which would be fatal to it in the active state. Encystment occurs in the free-living forms as the result of a gradual drying up of the water in which they live. In the parasites it is brought about upon expulsion to the exterior in the excreta of the host. Within its protective cyst wall the protoplasm may be wholly inactive or, more usually, divides into spores.

Ecologically, parasitic Protozoa may be divided into two groups, (*a*) parasites of the cavity or tissue of the alimentary canal, liver, kidney, and other organs, and (*b*) parasites of the blood. In the former case the part of the host invaded is in direct communication with the exterior, and hence the parasites, after completing their cycle of development within the body and becoming encysted, reach the outer world along with the excreta of the host. Once here, it is necessary for the further propagation of the parasite that the cysts get upon the food or in the drinking water of other members of the host species, then to be swallowed and to start a new generation of parasites. In such cases as these the dissemination of the cysts, although wholly a matter of chance, may be effected in any one of a number of ways. The excreta, deposited upon the ground, dry, and the cysts are blown about by the wind; the cysts may get into streams of water and thus be carried long distances; animals contaminate their skins and feet with moist feces, and thus carry the cysts. In addition to these purely natural methods, the cysts are doubtless often carried considerable distances in the course of commercial transactions; for example, by railroads.

Nevertheless, this mode of distribution is by no means so efficient as that occurring in the case of the protozoan parasites in the blood. These have wholly lost the power to encyst, and are altogether incapable of living in free nature. Hence the entire life is spent in the interior of some other animal, and these parasites are conveyed

from one vertebrate host to another by means of some biting invertebrate. It is evident that when this invertebrate is a flying insect, the parasite may easily be carried far and wide. In more detail, in cases where the parasite upon leaving the host merely falls to the ground, the infection may easily be confined to one pasture or stable or kennel. But when it is taken from the host by a biting fly it may be carried to neighboring pastures, stables, or kennels. In the former case the infection tends to be confined to within one circle, having for its center the point where the infection originally broke out, but in the latter each fly which so infects itself is the center of a circle of infection, and any animal within the range of its flight may become infected; hence there is not one but a great number of circles arranged in a group around the original focus of infection.

GROUPAL DIVISIONS OF THE PROTOZOA.

According to various peculiarities in their structure and mode of life, the Protozoa are zoologically grouped into five classes, as follows: (1) Spirochætida, (2) Flagellata, (3) Rhizopoda, (4) Sporozoa, (5) Ciliata.

Of these, the Sporozoa are exclusively parasitic, while both spirochætes and flagellates contain a large number of parasitic forms. The remaining groups, the rhizopods and ciliates, are nearly all free-living forms.

SPIROCHÆTIDA.

A spirochæte (fig. 63) consists of a long delicate body which ordinarily maintains the form of a corkscrew, hence the name. An undulating membrane, an extremely thin, finlike membrane running along the length of the body and following the curves, has been demonstrated to be present in some species. Delicate filaments or flagella may be present at one or both ends. The nuclear material instead of being massed together is apparently distributed throughout the length of the body in a series of granules.

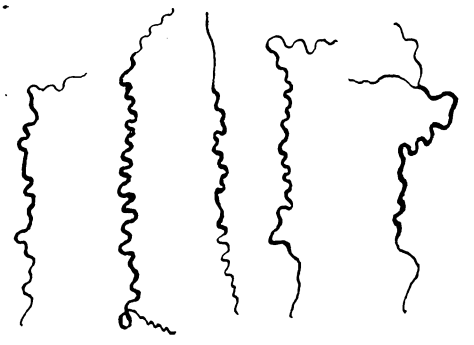


FIG. 63.—*Spirochæta pallida*. (From Doflein after Schaudinn.)

Spirochætes are motile organisms, frequently showing a rapid rotatory movement which may result in progression in either direction. They obtain their nutriment by osmotic absorption from the fluid in which they live. They produce by dividing lengthwise into two. This fact of longitudinal division is the principal reason

for regarding the spirochætes as Protozoa, rather than as Bacteria, since the latter, when reproducing by division, always divide transversely. In size spirochætes range from 1 micron to as much as 200 microns in length. Hence the smallest are among the most minute forms of animal life, while the largest might perhaps be seen with the unaided eye. So far as known the disease-producing species are nearer the lower limit than the upper limit of size.

The parasitic spirochætes with reference to their mode of life may be classified under five headings, as follows:

1. Inhabitants of the body cavities.
2. Inhabitants of normal mucous surfaces.
3. Inhabitants of inflamed mucous surfaces and ulcers.
4. Tissue parasites.
5. Blood parasites.

This series shows very clearly the way by which the origin of parasitism may be explained from the standpoint of evolution. Beginning with the inhabitants of the organic cavities, such as the intestine, spirochætes live merely as harmless commensals in the contents of these cavities, subsisting upon the food of the host rather than upon its secretions or tissues. The next step is the acquisition of the habit of living on the surface of the mucous membrane, in which case it may be assumed that the parasite subsists upon mucus. A further stage is reached by those forms which live in ulcers or degenerating tissues. The effete products resulting from the decay of the tissues furnish the spirochætes with an abundant supply of food, and in such regions they will multiply with great rapidity.

When the habit of living in ulcers and in tissues in process of breaking down is once acquired, it is an easy step to acquire the power of penetrating healthy tissues, and then of coming to dwell in the more deeply lying parts of the organism. Evidently the multiplication of spirochætes in such situations will cause local disturbances and the destruction of the invaded tissues. The effete products resulting from such tissue destruction, together with those coming from the bodies of such parasites as die, form toxins which, getting into the blood, are the cause of general symptoms.

The final stage is reached when the spirochætes acquire the habit of living in the blood. They then work harm to the organism by the production of toxins, which result from the dissolution of dead parasites in the blood, doubtless combined with substances produced by the parasites in the course of their metabolism.

In the case of the spirochætes which live in the blood, it is evident that transfer can not be by contact, but that the intervention of an intermediate host is necessary. This intermediate host must be a blood-sucking invertebrate, and in the known cases it is a biting bug or a tick.

In the present state of our knowledge the spirochætes do not seem to be so successful in causing disease in the domesticated animals as are other groups of Protozoa. Although occurring in cattle, horses, and sheep, the diseases produced are by no means severe and seldom result in death. With birds, chickens, and geese the diseases are of a much more severe type and show a large lethality. Even here, however, they are not of general occurrence and do not seem to cause any great loss. Yet the extreme pathogenicity of the spirochætes which are parasitic in man, namely, those causing syphilis, yaws, recurrent fever, and African tick fever, indicates that the group is a dangerous one to all the higher forms of animal life.

Hence it may well be that the spirochætes do much more harm than is supposed. There are a number of distinct diseases of domesticated animals for which the cause is unknown, and furthermore, many animals die of afflictions the symptoms of which are obscure or ill defined. In all such cases it is wholly within the limits of the possible that a spirochæte is to blame.

The following species of spirochætes may be mentioned:

SPIROCHÆTA ANSERINA.

This species is from 10 to 20 microns long, has a very delicate body, and many spiral curves. It occurs in geese, in which it causes a very fatal disease. It may be artificially transferred to chickens and ducks, in the young of which it causes a severe disease. During the incubation period, *S. anserina* lives in the spleen and bone marrow of the goose, later in the blood. The carrier of this species is not known.

SPIROCHÆTA GALLINARUM.

This species is 15 to 20 microns long. Discovered originally in Brazil, it occurs naturally in chickens, but is easily transmitted to geese, pigeons, ducks, and a number of sparrowlike birds. It can not live in mammals. One attack renders chickens immune. This spirochæte is remarkable in that it sometimes penetrates the red blood cells. It has also been found in the epithelium of chick embryos and in the eggs. The carrier is a tick, *Argas miniatus*.

A disease occurs among chickens in the southern United States which shows the same symptoms as the spirochæte disease. Furthermore, it seems always to be accompanied by the presence of the tick *Argas miniatus*, which has been proved to be the carrier of *Spirochæta gallinarum*, so that it is highly probable that the disease is a spirochætosis. Plate XXXVII, figure 1, shows a hen suffering from an infection of *S. gallinarum*.

SPIROCHÆTA THEILER

This species, discovered by Theiler in South Africa, measures 20 to 30 microns long by $1\frac{1}{4}$ to $1\frac{1}{3}$ microns wide. It is pointed at both ends.

It occurs in cattle and is transmitted by the tick *Boophilus decoloratus*. It is pathogenic, but only slightly so, seldom or never causing death.

SPIROCHÆTES OF HORSES AND SHEEP.

Spirochætes have been found in horses and sheep, but little is known concerning them or concerning the diseases which they may cause.

FLAGELLATA.

The Flagellata are rounded or elongated in shape, and are provided with one or more flagella. The form of the body varies a good deal (see Pl. XXXVIII and figs. 64 to 72) and is to a large extent correlated with the number and mode of insertion of the flagella.

The flagella, the characteristic organs of this group, are delicate, whiplike outgrowths from the body, capable of movement in any direction and of the most varied character. In number they range from one to six, or even more. Mostly they project freely from the body, but in some a flagellum forms the border of a delicate membrane running along the body and known, on account of its movements, as the undulating membrane. The flagella which form the border of the undulating membrane may be as long as the undulating membrane, or longer, projecting free.

Flagellates feed in various ways. Some of the free-living forms possess a mouth and cytopharynx, and live on solid food. But the greater number live by osmosis, and this is necessarily the method of nutrition of all the parasitic forms.

The parasitic genera of flagellates are as follows:

Trypanosoma (Pl. XXXVIII).
Cryptobia (=*Trypanoplasma*) fig. 64; Pl. XXXVIII, fig. 16).
Cercomonas (fig. 65).
Herpetomonas (fig. 66).
Crithidia (fig. 67).
Bodo (figs. 68 and 69).
Costia (fig. 70).
Monocercomonas.
Trichomonas (fig. 71).
Hexamitis.
Lamblia (fig. 72).
Polymastix.

As was seen to hold for the spirochætes, there is here also a regular gradation from harmless inhabitants of the cavities of the body up to the extremely pathogenic dwellers in the blood. A large number of forms live in such places as the recta of frogs and salamanders and in the various cavities of fresh-water animals. These, it may be assumed, are just beginning a parasitic life; the character of their

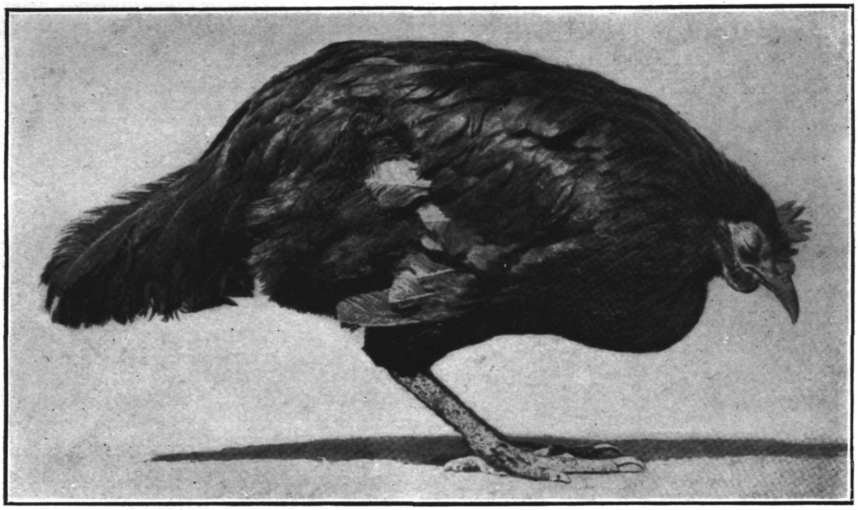
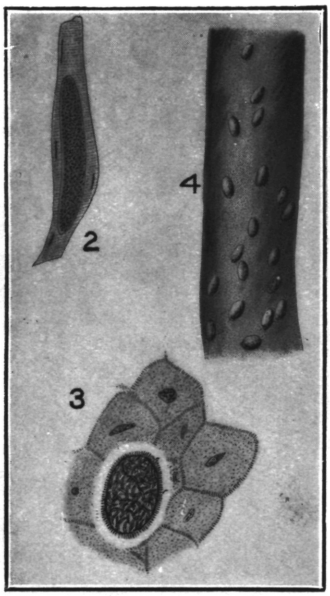
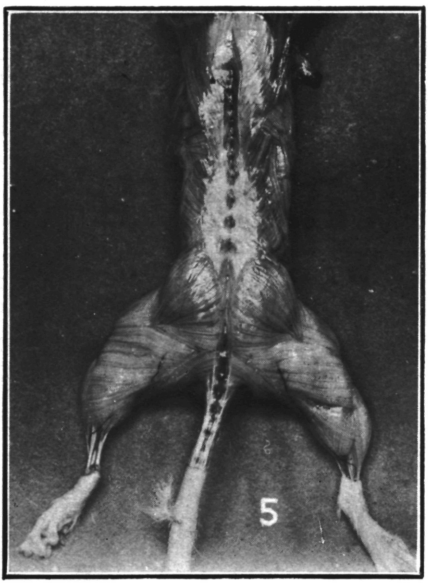


FIG. 1.—A HEN SUFFERING FROM ACUTE SPIROCHÆTOSIS. (AFTER BALFOUR.)





FIGS. 2 TO 5.—VARIOUS FORMS OF SARCOSPORIDIA.

2. *Sarcocystis blanchardi.* Longitudinal section of an infected muscle with young individual. (From Doflein after Van Eecke.)
3. *Sarcocystis tenella* in a Purkinje cell of the heart of a sheep. (From Doflein after Schneidemühl.)
4. *Sarcocystis tenella* in the wall of the esophagus of a sheep. (From Doflein after Schneidemühl.)
5. *Sarcocystis muris* in muscles of mouse.

environment differing but little from that of infusions of organic matter. Such parasites can scarcely be considered as doing any harm to their host, and presumably have selected such an environment on account of its furnishing a constant supply of food.

Other forms (*Trichomonas*, *Bodo*, *Lamblia*, etc.) live habitually in the alimentary canals of the higher animals. These are at times associated with morbid conditions and have then been noticed to become more abundant. It is not, however, supposed that they cause the morbid conditions. Rather it is the other way about, the morbid conditions furnishing a richer and more abundant pabulum which causes a more abundant growth of the parasite.

Finally, in the blood forms belonging to the genus *Trypanosoma* we come to some of the most destructive parasites known.

TRYPANOSOMA.

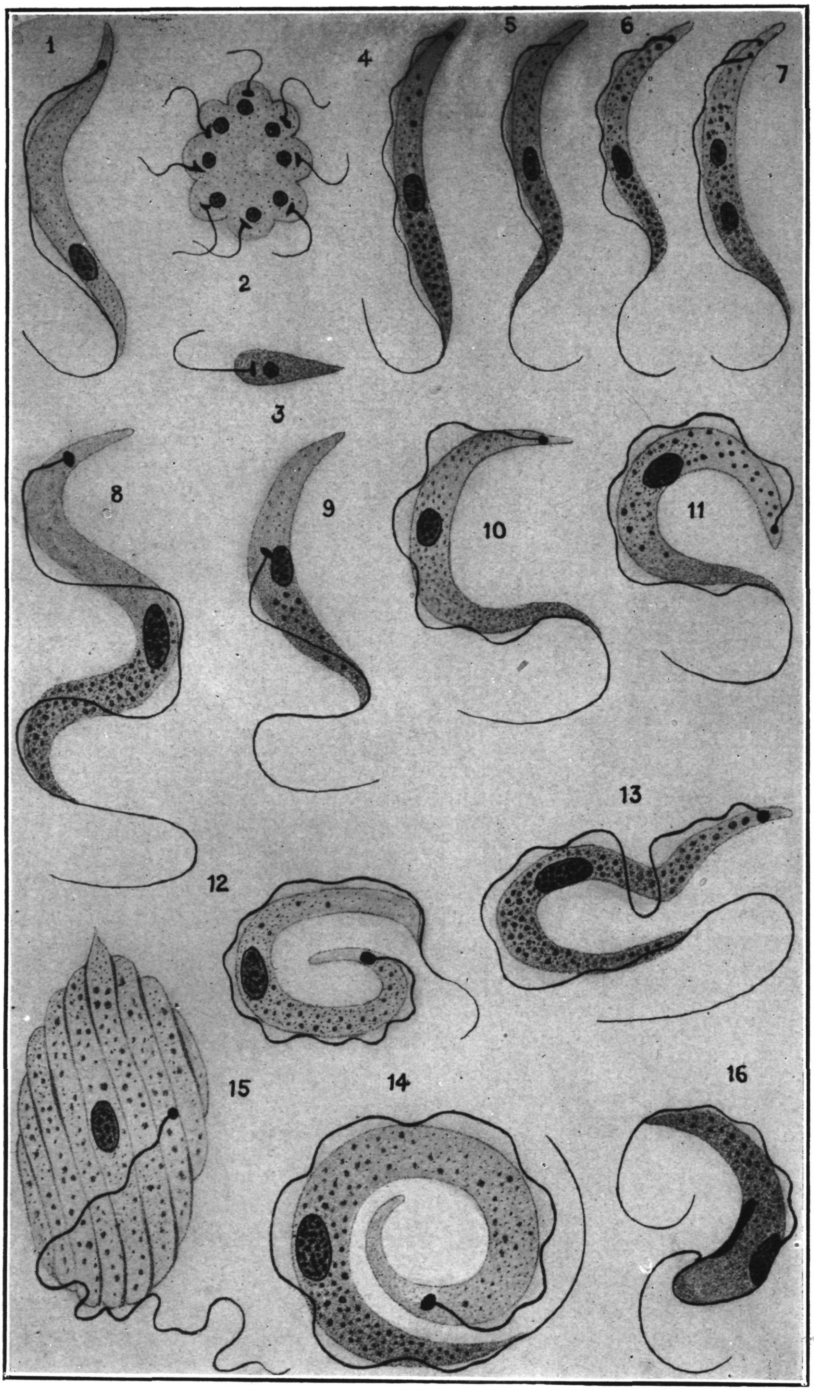
The form and appearance of the trypanosomes are shown on Plate XXXVIII. The body is elongated and may be blunt or pointed at the ends. Centrally, there is a round or oval body, the nucleus proper, or trophonucleus. Behind this, and usually very close to the posterior end of the body, is a very much smaller body, the centrosome, blepharoplast, or kinetonucleus. From the kinetonucleus arises the flagellum which runs to the surface, emerges, and passing forward forms the border of the undulating membrane. In most trypanosomes the flagellum is longer than the undulating membrane, displaying a free portion which may be as long as the entire body. It is conventional to refer to the end from which the free flagellum protrudes as the anterior end.

Trypanosomes are parasites of vertebrates ranging from fishes to man. They are confined to the blood, lymph, or cerebrospinal fluid, and hence, with one exception to be noted below, the intervention of an intermediate host is necessary for their transfer. In the case of certain of the mammalian trypanosomes the carrier is known to be a fly, and some biting arthropod is probably the intermediate host for the trypanosomes of all terrestrial vertebrates. In all cases where the carrier of the trypanosomes of fishes and frogs has been discovered it is some species of leech.

The transfer of trypanosomes by the invertebrate host is effected in two ways—the indirect or biological and the direct or mechanical. The former is wholly analogous to what takes place in the transmission of malaria by mosquitoes. The fly bites an animal suffering from the disease, taking up a number of trypanosomes with the blood. These are not digested by the fly, but go through certain biological processes, and may apparently even disappear as trypanosomes. At all events the fly, after the first few hours, generally ceases to be

PLATE XXXVIII.—VARIOUS SPECIES OF TRYPANOSOMA.

1. *Trypanosoma lewisi*, of the rat.
2. *Trypanosoma lewisi*, multiplication rosette.
3. *Trypanosoma lewisi*, small form resulting from the disintegration of a rosette.
4. *Trypanosoma brucei*, of nagana.
5. *Trypanosoma equinum*, of caderas.
6. *Trypanosoma gambiense*, of sleeping sickness.
7. *Trypanosoma gambiense*, undergoing division.
8. *Trypanosoma theileri*, a harmless trypanosome of cattle.
9. *Trypanosoma transvaliense*, a variation of *T. theileri*.
10. *Trypanosoma avium*, a bird trypanosome.
11. *Trypanosoma damoniæ*, of a tortoise.
12. *Trypanosoma soleæ*, of the flat fish.
13. *Trypanosoma granulosum*, of the eel.
14. *Trypanosoma rajæ*, of the skate.
15. *Trypanosoma rotatorium*, of frogs.
16. *Cryptobia borreli*, of the red-eye (a fish).



VARIOUS SPECIES OF TRYPANOSOMA. (FROM LAVERAN AND MESNIL.)

infective, and remains noninfective for a number of days. After this lapse of time, however, the trypanosomes within the fly resume their ability to infect any host which the fly may bite. Moreover, flies which have thus become infective remain so, so far as is known, for the remainder of their lives, the trypanosomes continually multiplying within them. In the experimental work so far done, however, only a small proportion of flies—from 5 to 20 per cent—acquire this permanent infection, although it is believed that in nature this percentage is much larger.

The second method of transfer is the direct or mechanical. If a fly bites a sick animal, and very shortly afterwards a healthy one, the latter may contract the disease. This is due to the fact that as a result of the first bite the proboscis of the fly becomes charged with trypanosomes and these are deposited in the wound made when the fly bites for the second time. The experiment has been tried of permitting a fly to bite a sick animal, and then, successively, two healthy ones. It was found that of the latter the one first bitten usually contracted the disease, whereas the second did not. In other words, the fly cleaned its proboscis while biting the first of the healthy animals. The ability to infect by the direct method is usually lost at the end of a few hours, but has been maintained for as long as two or three days.

In the transfer of a trypanosome by the indirect method, while there may be more than one insect host, these, in the known cases, are closely allied species. This is what we should expect, as it is unlikely that a protozoan, adapted to spend a portion of its life cycle in a fly, could do so equally well in a bug or a tick. This, moreover, is confirmed by observations made on the transmission of the trypanosomes of cold-blooded vertebrates by leeches. Here it has been found that a given species of leech is necessary. In other than the right leech the trypanosomes live for a time, but can not go through the proper developmental stages, and such leeches do not become infective.

In direct transmission it apparently makes but little difference what species of biting arthropod is involved, the essential condition being that the interval between bites be only a matter of a few hours. The fly acts merely as a scalpel or hypodermic needle. Indeed, it has been found that the house fly, which can not bite at all, can transfer surra by sucking alternately a raw surface on a sick and a well animal.

In the blood of vertebrates trypanosomes increase by bipartition, which is always longitudinal. It may be a mere splitting into two generally equal daughter cells, or a sort of rosette formation (see Pl. XXXVIII, fig. 2), which latter, however, is only bipartition in which the daughter cells remain together for a longer or a shorter

period of time. Nothing in the way of conjugation or encystment has ever been observed.

Knowledge as to what takes place in the intermediate host is meager. Here, in addition to normal trypanosomes, there are flagellates resembling the more primitive cercomonads or herpetomonads. These are presumed to arise from division of the imbibed trypanosomes, but the exact processes taking place in the fly have not yet been worked out.[1]

TRYPANOSOMA GAMBIENSE.

This is the agent of sleeping sickness, and while primarily of interest to human medicine, is also of interest to the live-stock industry, since it has been found to be pathogenic for many other animals besides man. Sleeping sickness seems to have been first described by Winterbottom, in 1803, as occurring in Sierra Leone, on the West Coast of Africa. It was not, however, until a good deal later, from 1860 onward, that it began to attract attention. Between 1891 and 1902 a considerable number of alleged "causes" were discovered, all belonging to the bacteria. A blood filaria was also blamed, and one or two investigators attributed the disease to errors in diet. In 1901, however, Dutton, examining the blood of a European patient at Bathurst, Gambia, recognized the parasite as a trypanosome and named it *Trypanosoma gambiense*. (See Pl. XXXVIII, figs. 6, 7.)

A little later Castellani found a trypanosome in the cerebrospinal fluid of negroes in Uganda. It had not at first been known that the Uganda disease and that of the West Coast were the same, but the discovery of trypanosomes in both suggested their identity, a conclusion which has been confirmed by all subsequent work.

Sleeping sickness, then, ranges from the West Coast of Africa across to Uganda, and as a result of the recent exploitation of this region, is spreading. At present its range is the valleys of the Senegal, Niger, Congo, and Upper Nile Rivers.

The disease shows two phases, the first that of so-called trypanosome fever, the second that of sleeping sickness. The first stage, which may last from a few weeks to several years, is characterized by enlargement of the glands and generally by fever. The trypanosomes are always scanty, and can, as a rule, be found only in gland juice. In the second stage, or sleeping sickness, the marked feature is the gradually increasing lethargy. I quote from the English edition of Laveran and Mesnil, page 374:

> The drowsiness increases, and the patient's attitude becomes characteristic. The head falls forward on the chest and the eyelids are closed. At first the

[1] Very recently, however, it has been shown that in the tsetse fly, *Trypanosoma gambiense* does not undergo any radical morphological changes, and that after remaining for a certain time in the alimentary canal it gains the salivary glands.

patient is easily aroused from this drowsy condition, but soon he reaches that stage in which he falls sound asleep, almost in any attitude, and under any conditions, especially after meals. These periods of sleep, which become gradually longer and more profound, lead eventually to a comatose condition, from which the patient can be aroused only with the greatest difficulty. It is at this stage that the temperature becomes subnormal and death occurs.

Sleeping sickness is ordinarily fatal, although occasional cures are reported. Besides man, *Trypanosoma gambiense* is pathogenic for a large number of other mammals—monkeys, lemurs, the dog, jackal, cat, rabbit, guinea pig, rat, mouse, jerboa, hedgehog, marmot, horse, donkey, cow, goat, and sheep.

The carrier is *Glossina palpalis*, a species of tsetse fly. Experimental work shows that the fly, after biting an animal harboring *T. gambiense*, is noninfective for a period of some days, after which it becomes infective. There are also reasons for suspecting that other species of *Glossina* can carry sleeping sickness.

TRYPANOSOMA BRUCEI.

In 1895 Bruce discovered that nagana, or the so-called tsetse-fly disease of Africa, was caused by a trypanosome. He writes:

> Nagana, or the fly disease, is a specific disease which occurs in the horse, mule, ox, dog, cat, and many other animals, and varies in duration from a few days or weeks to many months. It is invariably fatal in the horse, donkey, and dog, but a small percentage of cattle recover. It is characterized by fever, infiltration of coagulable lymph into the subcutaneous tissue of the neck, abdomen, or extremities, giving rise to swelling in these regions, by a more or less rapid destruction of the red blood corpuscles, extreme emaciation, often blindness, and the constant occurrence in the blood of an infusorial parasite.

Bruce adds that nagana is a Zulu word, and has reference to the state of depression and weakness of the infected animal.

The disease is present generally throughout Africa north of the Tropic of Capricorn, except Tunis, Algeria, and Morocco. As in the case of sleeping sickness, the trypanosome can live in nearly all mammals, and to most of them it is fatal. In rats and mice division proceeds with such energy that the parasites may become as abundant as the red blood cells. The parasite, which is appropriately given the name of its discoverer, is shown in figure 4 of Plate XXXVIII. The carrier is not certainly known, the credit generally being given to *Glossina morsitans*, but *Glossina pallidipes* is regarded as playing perhaps an equal rôle in this respect. As yet, however, no accurate experimental work has been undertaken with these flies.

TRYPANOSOMA EVANSI.

Surra plays the same part in Asia which nagana does in Africa. Surra is a Hindu word meaning rotten. The parasite causing the disease, *Trypanosoma evansi*, was discovered by Steel in 1885. The

disease occurs naturally in horses, camels, and dogs, and is inoculable into a large number of animals with the usual fatal results. Horses always die, the duration of the disease being from a few weeks up to six months. Camels resist the disease for as much as three years. In India cattle are generally resistant, although harboring the parasite in their blood. When, however, in 1901, the disease was introduced into the island of Mauritius cattle succumbed as easily as horses.

The disease occurs throughout southern Asia from Persia to China, in all of the East Indian islands, the Philippines, Korea, Australia, and in Africa in Mauritius and in Madagascar. Camel surra, under the native name of mbori, also occurs in northern Africa.

The specific carrier is unknown. The tsetse flies are not known in Asia, their place being taken by Tabanids and the ubiquitous *Stomoxys calcitrans*, the stable fly. It has been determined that surra can be transmitted by any or all of these flies, but so far as the evidence goes, none of them are infective for more than a day or so after biting an infected animal, and they thus serve merely as direct carriers.

TRYPANOSOMA EQUINUM.

Mal de caderas, or, more briefly, caderas, caused by the parasite *Trypanosoma equinum* (see Pl. XXXVIII, fig. 5), is a disease which affects horses in South America. The following is taken from the English edition of Laveran and Mesnil, page 293:

> The first sign of the disease in horses is wasting, which rapidly progresses in spite of a good appetite. The temperature is often raised to 104° to 105.8° F. After a variable time it is noticed that the hind quarters are weak, and that the animal drags its legs, the hoofs grazing the ground. These symptoms increase and become characteristic, so that when the animal is made to walk it staggers along, the hind quarters swaying from side to side. On account of this symptom the name mal de caderas, or disease of the hind quarters, has been given to the disease. There comes a time when the animal is unable to stand. If in the stable it leans up against the wall or seeks other support, if in the open it staggers and falls. After thus falling to the ground an animal may live for several days if it be fed; otherwise the inevitably fatal end is hastened by inanition.

The parasite, *T. equinum*, was discovered in 1901 by Elmassian. The only domesticated animals in which caderas occurs spontaneously are horses and, rarely, dogs. But the carpincho, a large rodent, is very susceptible, frequently dying in large numbers, and such outbreaks are frequently the forerunners of epizootics among horses.

The disease ranges throughout the greater part of South America, but is not present in the northwestern part of the continent, nor along the Pacific coast.

Caderas is always fatal to horses, and also to a considerable number of small animals to which it may be artificially inoculated. It has,

however, little or no effect on cattle, sheep, goats, and swine. If such animals be artificially inoculated, the trypanosomes may remain present in their blood in very small numbers for a longer or shorter time, but no inconvenience is suffered.

TRYPANOSOMA EQUIPERDUM.

Dourine, maladie du coït, or horse syphilis, is a disease of horses which is present more or less throughout the world. It is, however, by no means so important to veterinary science as are other animal trypanosomiases. This disease is peculiar in that there is no intermediate host, the trypanosome being inoculable by contact, like the spirochæte of human syphilis. Infection is usually by coition, and hence only stallions and brood mares are involved.

It was originally supposed that the trypanosome could gain entrance only through abrasions in the mucous membranes, but recent work shows that it is able to work through wholly intact membranes. In general, dourine runs a chronic course, lasting from two to six months, sometimes as long as two or three years. Occasionally it is acute, killing the animal within a shorter period.

Dourine is inoculable to dogs, rabbits, and other animals, but is not pathogenic for as many species as are nagana, surra, or caderas.

Infection being by contact, dourine is easily controlled, since all that is necessary to do is to destroy the infected animals. The problem presents none of the difficulties that come up in the case of diseases like nagana and surra, where not only must the diseased animals be destroyed, but all healthy ones must be shielded from the attacks of possibly infected flies.

TRYPANOSOMA AMERICANUM.

This species, though apparently a harmless parasite, deserves attention on account of its common occurrence among American cattle. Its presence in cattle can usually be demonstrated only by the culture method, the trypanosomes, if present, becoming sufficiently numerous in the tubes in a few days to be found readily by the microscope. It occurs in the United States and also in the Philippine Islands and in Europe.

OTHER TRYPANOSOMES.

Trypanosoma dimorphon causes a specific disease of horses in Gambia and in French Guinea. The carrier is *Glossina palpalis*.

Trypanosoma vivax attacks cattle, sheep, and goats in the coast region of the Cameroons. *Trypanosoma congolense* is pathogenic for sheep and pigs in the Congo Free State.

Other trypanosomes might be mentioned which attack domesticated animals in Africa, where every mammal seems to have several

parasites bent upon its destruction. It would, however, be only tedious to give a list of all of them.

TRYPANOSOMES OF BIRDS, REPTILES, BATRACHIA, AND FISHES.

In birds trypanosomes are of wide distribution, but generally rare in the individual bird. Practically nothing is known as to their pathogenicity. A French savant, Thiroux, by artificially inoculating Java sparrows with trypanosomes which came from other birds of the same species in some cases produced death. But, so far as our knowledge goes at present, the trypanosomes of birds are of little economic interest.

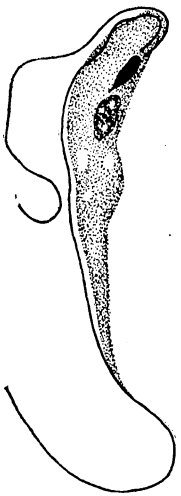


FIG. 64. — *Cryptobia helicis*. (From Bureau of Animal Industry Bulletin 119.)

Reptiles and batrachians are frequently infected with trypanosomes, but the disease of these animals are not of interest in the present connection.

A large number of both fresh-water and marine fishes are parasitized by trypanosomes. Following, however, what seems to be the usual rule for vertebrates lower than mammals, the organisms are generally scanty in the blood. There is no good evidence that any member of the genus *Trypanosoma* is pathogenic for fishes, yet it would be a leap in the dark to say that none is. So far, the fish trypanosomes have been studied wholly from the zoological viewpoint, the question of their pathogenicity not being taken into account.

FIG. 65.—*Cercomonas crassicauda*. (After Doflein.)

CRYPTOBIA.

The genus *Cryptobia*, usually known as *Trypanoplasma*, was created in 1846 by Leidy for an organism living in the seminal vesicles of a snail. The members of this genus look a good deal like trypanosomes, but differ in two respects. They are portrayed in figure 64 and in Plate XXXVIII (16). From these it may be seen that there are two flagella, one forming the edge of an undulating membrane, the other projecting freely. In addition, the kinetonucleus, instead of being a small granule, as in trypanosomes, is here a body which may be nearly as large as the trophonucleus. It is the convention to regard the thicker end—the one from which the free flagellum projects—as anterior, although this appears to reverse the relations as they are interpreted in the trypanosomes.

Cryptobia is not in all cases a blood parasite. The type species, as stated, lives in the seminal vesicles of a snail, and another, *Cryptobia intestinalis*, is an intestinal parasite. Mostly, however, the members of the genus are parasites of the blood of fishes, and some are known to be highly pathogenic. So far as is yet known, no species of this genus attacks any of the domesticated animals.

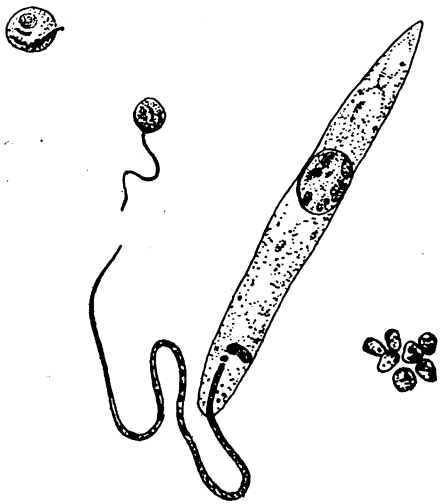


FIG. 66.—*Herpetomonas jaculum*. (After Porter.)

CERCOMONAS.

The appearance of this organism is indicated by figure 65.

A number of flagellated parasites have been credited to the genus *Cercomonas*, but in the main they are poorly described. They occur in the intestines of invertebrates, and of man, the dog, goose, and fowl. Cercomonads have also been said to occur in wounds and ulcers, but in some cases, at least, it is questionable if such observations are authentic. Little can be said as to the pathogenicity of the members of this genus.

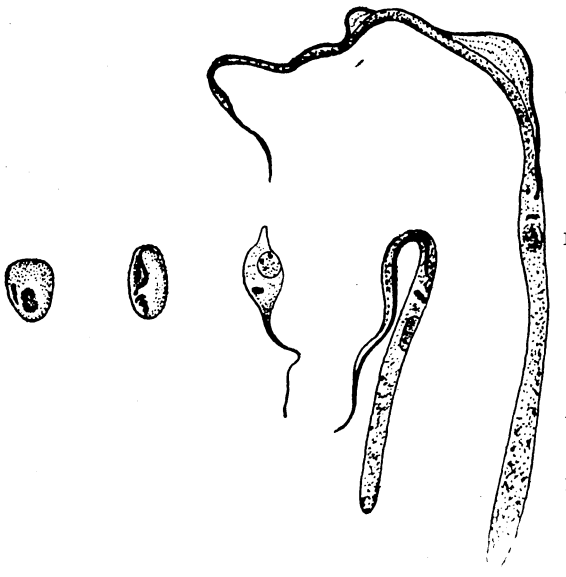


FIG. 67.—*Crithidia gerridis*. (After Porter.)

HERPETOMONAS.

The species of this genus (see fig. 66) are elongated flagellates with a long flagellum at the anterior end. There is a kinetonucleus, from which arises the flagellum, and a trophonucleus. All are parasites, and so far all have been found in the intestines of invertebrates. They are common in flies and mosquitoes.

CRITHIDIA.

Crithidia (fig. 67) is a genus which stands between *Herpetomonas* and *Trypanosoma*. There is a flagellum, a short undulating membrane, a kinetonucleus, and a tropho-

nucleus. The species described are parasites of insects and hence of no special interest in the present connection. In their evolution in culture tubes trypanosomes pass through a crithidial stage, suggesting that *Trypanosoma* has, in its evolution, passed through a *Crithidia* stage.

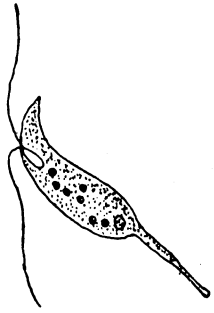


FIG. 68.—*Bodo grylotalpæ*. (From Doflein, after Grassi.)

BODO.

Bodo (figs. 68 and 69) is a biflagellate organism, bearing a certain resemblance to *Cryptobia*. This comes about from the fact that whereas *Bodo* has no undulating membrane, one of the flagella is usually bent backward and is kept constantly close to the body. There are also, as in *Cryptobia*, two nuclei. All the species of *Bodo* are parasites, mostly of invertebrates. One, *B. urinarius*, has been observed in freshly discharged human urine.

FIG. 69.—*Bodo lacertæ*. (From Doflein, after Prowazek.)

COSTIA.

Costia (fig. 70) possesses three flagella according to some authorities, or four, according to others. The genus is important in that it does considerable damage as a parasite of food fish. But *Costia* differs from all of the other flagellates which have been considered because it is an ectoparasite and lives on the skin and gills. Goldfish, trout, trout embryos, carp, and other fish are attacked. The parasite anchors itself with its long flagellum, becoming so firmly attached that it can be removed only with difficulty.

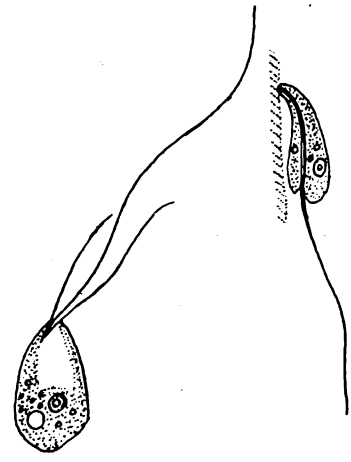


FIG. 70.—*Costia necatrix*. (From Doflein, after Henneguy.)

These parasites may be present in enormous numbers. They provoke lesions of the skin and hemorrhages of the gills. Freshly infected embryos die in two days; goldfish live longer, but slowly succumb. No successful treatment has ever been discovered.

MONOCERCOMONAS.

This is a small form possessing four flagella. The species are all parasites, living in the intestines of vertebrates and invertebrates. There is no good reason to regard any as pathogenic.

TRICHOMONAS.

This genus (fig. 71) is characterized by the possession of either three or four equally long, free flagella, arising at the anterior end. In those cases where only three flagella are present there arises from the same point of origin an undulating membrane which runs spirally around the body and often is prolonged behind into a free flagellum.

FIG. 71.—*Trichomonas batrachorum.* (From Doflein, after Blochmann.)

Trichomonads occur in batrachia, lizards, snakes, birds, and in many mammals. The various forms occurring in these different hosts are all much alike morphologically, and it is not known whether there are actually few or many distinct species. They are mostly parasites of the alimentary canal, but one form is at times found in the vaginal mucus of *Homo*. Whereas they are at times associated with morbid conditions, there is no reason to suppose them pathogenic.

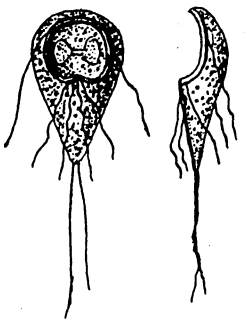


FIG. 72.—*Lamblia intestinalis.* (From Doflein, after Grassi and Schewiakoff.)

HEXAMITUS.

The species of the genus *Hexamitus* are in some cases intestinal parasites, but are apparently not of much economic importance.

LAMBLIA.

Lamblia (fig. 72) lives in the intestines of man and other mammals. In inflammation of the intestinal mucous membrane *Lamblia* becomes very much more abundant, and recent work suggests that such great increases in numbers may cause dysentery. It may be noted also that *Lamblia*, unlike *Trichomonas*, *Bodo*, etc., spends its life upon the mucous membrane rather than free in the intestinal conduits. As shown by the figure, the anterior end of the parasite is hollowed out to form a saucerlike depression, and the flagellate attaches itself to the free ends of the intestinal cells by fitting over them this saucerlike depression. It is accordingly easy to see that when present in large numbers the parasite, by forming a sort of coating on the mucous membrane, might set up disturbances.

RHIZOPODA.

The general appearance of a simple rhizopod is shown by figure 73, *Amœba proteus*, the common fresh-water ameba. It is seen to show a differentiation into ectosarc and endosarc, the difference between the two being that the ectosarc is somewhat more dense and does not contain granules. Amebas are organisms which continually undergo changes of shape, these changes constituting a type of motion sui generis, which when displayed by other Protozoa and by metozoan cells is spoken of as ameboid. Within the endosarc are the nucleus and contractile vacuole, permanent possessions, and in addition to these food vacuoles and granules of various sorts.

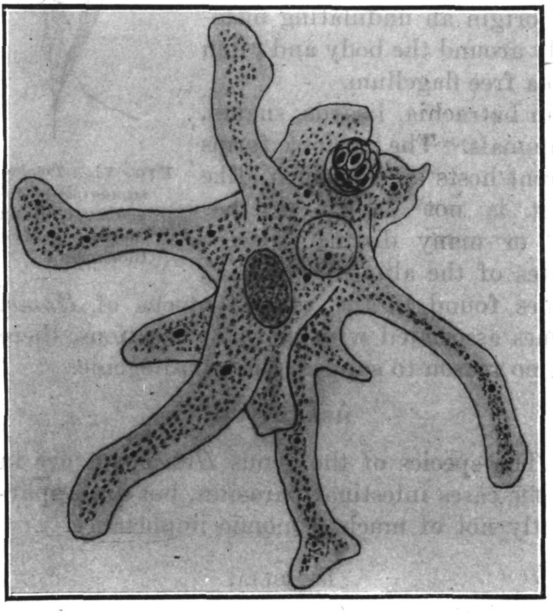


FIG. 73.—*Amœba proteus*. (After Doflein.)

Rhizopods seem always to subsist upon solid food, which is merely engulfed; that is, on coming into contact with a particle of food the ameba merely flows around and incloses it. Within the cytoplasm a certain amount of liquid is secreted around the food particle, thus forming a food vacuole. This liquid presumably holds some chemical in solution, since in the course of time the digestible portions of the food particle are liquefied and absorbed, while the residuum is cast out by a process much the same as that which brought it in.

The known parasitic species are few in number and belong to either one of the two genera, *Amœba* and *Entamœba*. Four species are known to occur in man, one of them the cause of a rather serious disease, amebic dysentery, which is widespread in the Tropics. Ameboid organisms are by no means rare in the alimentary canal of domesticated animals, but there does not seem to be any satisfactorily authenticated case of pathogenicity. *Amœba meleagridis* is the name given by Theobald Smith to an organism found in the liver and cæca of turkeys suffering from blackhead. It is not doubted that a parasite is present in the lesions caused by this disease, but its

identification as an *Amœba* is questionable. Another doubtful case is that of a fatal disease of sheep, occurring in Australia in 1885. This was credited to an *Amœba*, but the evidence furnished was not conclusive.

THE SPOROZOA.

The Sporozoa are all parasites, and considered as a group are the most dangerous of all disease-producing organisms. They parasitize all classes of animals, from other Protozoa upward, and have at times been recorded in man. The name, Sporozoa, has reference to the fact that these animals reproduce by the formation of spores, which are typically minute, boat-shaped, shelled structures, containing one or more smaller bodies, the sporozoites.

It is taken as an axiom that parasites are descended from free-living animals, and several views have been put forward regarding the ancestry of the Sporozoa. It is found, however, that a hypothesis which may seem plausible for one portion of the Sporozoa fails for the other portion, and hence protozoologists are disposed to believe that the Sporozoa are polyphyletic; that is, that different subdivisions have had different ancestral histories.

Zoologically, the Sporozoa may be divided into two divisions, Telosporidia and Neosporidia, and the somewhat more favored view is that the former are descended from flagellates and the latter from rhizopods. These two groups are distinguished in this way: In the Telosporidia there is a longer or shorter vegetative life, followed by conjugation. The product of conjugation, technically known as the zygote, divides up into spores, and ceases to exist as a zygote. But in the Neosporidia, spore formation is inaugurated quite early in the life history of the individual and vegetative growth and spore formation proceed together.

TELOSPORIDIA.

The only members of this group of interest in the present connection are the Coccidiomorpha, which zoologically are divided into the Coccidia and Hæmosporidia.

COCCIDIA.

The Coccidia are cell parasites, attacking tissue cells and especially epithelium, but never cells of the blood. They are parasites of arthropods, molluscs, and vertebrates. They are distinctly pathogenic for vertebrates, and are doubtless also usually pathogenic for anthropods and molluscs, but the diseases of these last-named animals are not of special economic interest and hence are scarcely known.

Infection of Coccidia is by way of the mouth. Hence, as a rule, it is the epithelium of the alimentary canal and its appendages which

PLATE XXXIX.—COCCIDIAN LIFE CYCLE.

Figure 1.

1. Sporozoite, released in intestine of host.
2. Penetration of sporozoite into epithelium cell.
3, 4. Growth of sporozoite into trophozoite.
5, 6, 7. Schizogonous cycle. Nuclear division, followed by division of entire trophozoite into a large number of merozoites.
8. Free merozoites, which for an indeterminate number of generations merely repeat the schizogonous cycle, behaving precisely as do the sporozoites. Eventually, however, the sporogonous cycle is initiated, which proceeds as follows:
9a. Undifferentiated female cell.
9b. Undifferentiated male cell.
10a. Differentiated female cell.
10b. Differentiated male cell.
11, 12. Formation of the microgametes, one male cell producing many microgametes.
13a. Macrogamete. One female cell produces but one macrogamete.
13b. Ripe microgamete.
14. Fertilization.
15, 16, 17. The zygote.
18. Beginning of spore formation.
19. Completion of spore formation.
20. Formation of the sporozoites within the spores.
21. Release of the sporozoites in the intestine of the host.

Figure 2.

Introduced for comparison with the more typical cycle shown in figure 1. Here the parasite penetrates and comes to rest in the nucleus instead of the cytoplasm, and there is sexual differentiation in the schizogonous cycle as well as in sporogony.

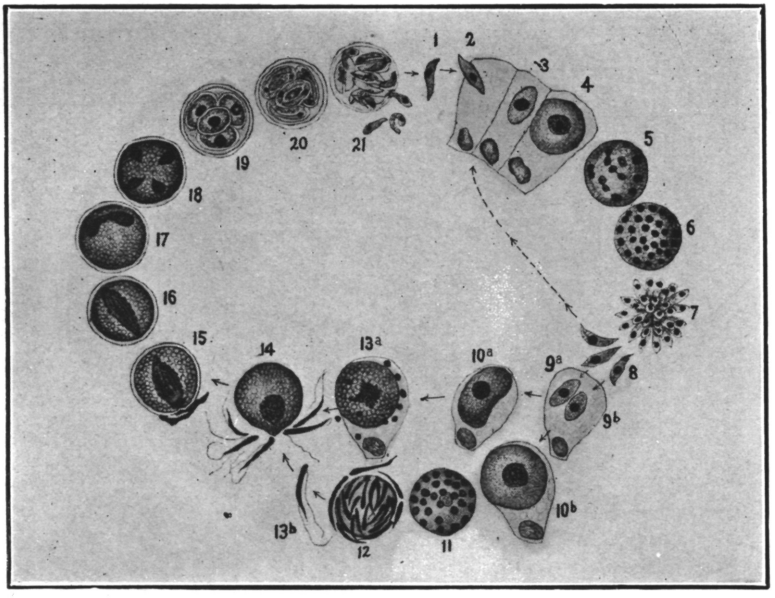


FIG. 1.

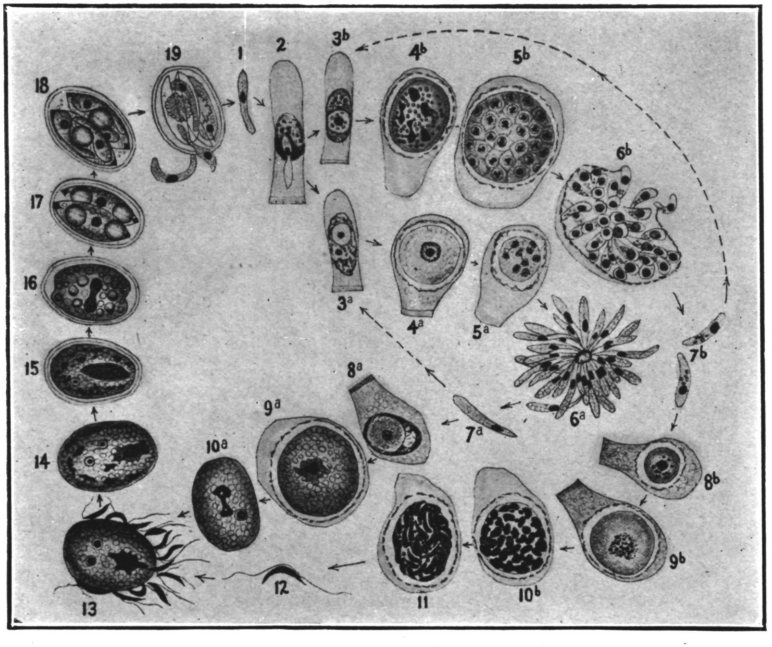


FIG. 2.

COCCIDIAN LIFE CYCLE. (FROM MENSE'S "HANDBUCH," AFTER SCHAUDINN.)

is attacked, but Coccidia also invade the kidney, spleen, or testis. The life history of a coccidian is as follows (see Pl. XXXIX):

The encysted stage, or oocyst, is swallowed by some animal. Through the action of the digestive juices the sporozoites contained in the cyst are released in the lumen of the intestine. Each sporozoite then enters an epithelial cell of the mucous membrane lining the intestine. The sporozoite may then either settle down in this cell and proceed to grow, or else it may pass entirely through the intestinal wall, and eventually come to rest within a cell of the kidney or testicle. In either case, once established, the organism, now termed a trophozoite, grows until it reaches a certain definitive size, which is probably primarily dependent upon the size of the invaded cell. Then by a process termed schizogony the trophozoite divides into a number of small masses of protoplasm known as merozoites. These migrate to and enter other cells of the host, and then grow and divide in turn into another generation of merozoites. As a result of this schizogonous cycle, which may be repeated many times, a sufficient number of cells may be destroyed to cause the death of the host animal. Whether or not the host is killed, there finally comes a time when some of the merozoites, instead of growing and dividing into other merozoites, develop into sexual stages and thus begin the process known as sporogony. The male trophozoite divides into a number of very minute elements, known as microgametes. The female trophozoite does not divide, but develops into a macrogamete, or egg. The microgametes are motile, and like the spermatozoa of higher animals, seek out and fertilize the eggs. Immediately after fertilization the egg surrounds itself with a protective covering and becomes an oocyst. The oocysts reach the exterior with the feces of the host in the case of those species which are parasitic in cells of the alimentary tract or its appendages. Probably in some cases the oocysts reach the exterior only as a result of the host's death and disintegration. But, however this may be, the protoplasm forms within the cyst one or, by division, several spores, each of which becomes inclosed in a shell, the sporocyst. The protoplasm within the sporocyst then divides into two or more sporozoites, and the sporogonous cycle is complete. Upon the swallowing of the oocyst the processes described above are again repeated.

It will be noted from the account which has been given of the life history of the Coccidia that the number of parasites which may be produced as a result of infection does not necessarily depend upon the number of cysts swallowed, but upon the number of schizogonous generations, or repetitions of the schizogonous cycle. Were it not for the fact that sooner or later the process of reproduction by schizogony comes to an end, the host would invariably be killed by the extensive destruction of the cells of the organ or tissue attacked by the parasites. Since, however, the number of schizogonous generations is limited—by what agencies we are as yet unaware—the infected animal tends to recover if it survives the acute stage of the disease during which the parasites are multiplying by schizogony. With the inauguration of the sporogonous cycle the destruction of the cells of the host ceases, the acute symptoms subside, and meanwhile, through the recuperative powers of the affected tissues, new cells have taken the place of those destroyed by the parasites, so that the injury to the host is more or less completely repaired. Apparently,

however, no immunity is produced, and another attack of the disease may result from a fresh infection from without.

Eimeria stiedæ.—This species, also known as *Coccidium oviforme*, causes a serious coccidiosis in rabbits, and though rare among wild rabbits, is a very common parasite of domesticated rabbits. It infests the epithelium of the bile ducts, causing, in addition to cellular destruction, enlargement of the liver and compression of its blood vessels. The secretion of bile is reduced, the blood becomes pale and watery, and the animal finally may become so profoundly affected that death results.

Forms similar to this species attack other small mammals, such as the mouse. Coccidiosis in man has been attributed to *Eimeria stiedæ* by some authors.

Coccidium zurni.—This coccidian is considered to be the cause of red diarrhea of cattle. The disease has been noted in Europe, where outbreaks of a similar character have also occurred among sheep. The lethality, however, for cattle seems to be rather low, varying from 2 to 4 per cent. As it is the intestinal cells which are attacked by the parasite, the mucosa of the intestine becomes stripped off in places, resulting in extensive hemorrhage into the lumen, which causes the red diarrhea. In fatal cases, death results within two days. Most of the recorded cases have occurred in Switzerland, in summer and autumn. Dampness favors the disease, probably for the reason that in dry weather the encysted infective stages of the parasite become desiccated, and thus many are killed.

Coccidia of birds.—Birds are very commonly infested with Coccidia. Investigations by this bureau[1] show that white diarrhea in chickens is caused by Coccidia and that intestinal coccidiosis of pigeons is also due to the same cause. Blackhead of turkeys has been ascribed by some investigators to a coccidian.

HÆMOSPORIDIA.

Like the Coccidia, the Hæmosporidia are cell parasites, but instead of being found in epithelial cells they occur in the cells of the blood, for the most part in the erythrocytes, or red corpuscles. The group contains some of the most dangerous parasites known, and is in this respect perhaps quite similar to the trypanosomes. In general, however, the diseases caused by Hæmosporidia do not have so high a rate of lethality as the trypanosomiases, but they attack a larger number of individuals. It is now a matter of common belief that one of the principal causes of the decadence of the ancient Greek and Roman civilizations was malaria, the most important hæmosporidian disease of man.

[1] Morse, George Byron. White diarrhea of chicks. U. S. Department of Agriculture, Bureau of Animal Industry, Circular 128. 1908.

The evident reason why the Hæmosporidia are more dangerous than the Coccidia is that they are not transmitted by contact or contamination, but by means of intermediate hosts. These, in the known cases, are mosquitoes, bugs, fleas, and ticks. The typical life history of the Hæmosporidia is as follows (see Pl. XL):

As in the case of the Coccidia, the hæmosporidian begins its career within its host as a sporozoite. Set free in the blood of the host, this sporozoite attacks and enters a blood cell, preferably an erythrocyte. Here it grows into a trophozoite, which is ameboid in one group of Hæmosporidia, the Acystosporea, and generally vermiform in another group, the Hæmosporea. The trophozoite, growing at the expense of the blood cell, soon breaks up into a number of merozoites. By the disintegration of the blood cell, the merozoites fall into the blood stream. Forthwith they attack new blood cells and the process (schizogony) is repeated. The increase is therefore by geometrical ratio, and, as Minchin says: "It is evident that reproduction at this rate could only continue indefinitely in the ichor of an infinite host." Accordingly, at the end of a certain number of generations, as in the Coccidia, schizogonous reproduction ceases and the parasite provides for its future by the production of male and female elements.

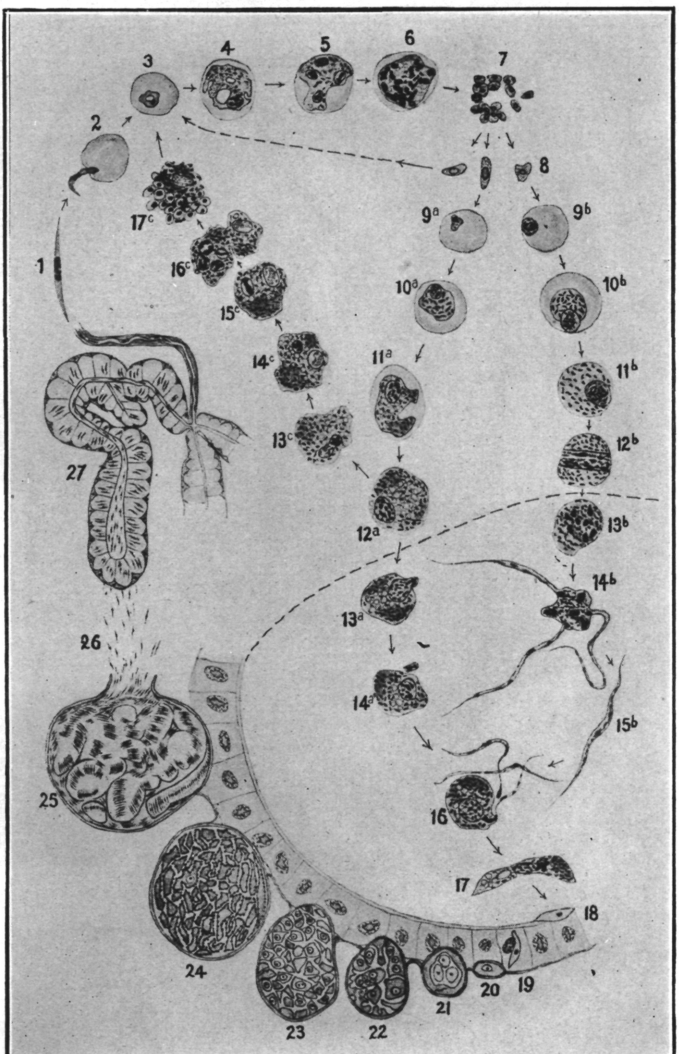
So far, the process is exactly parallel to that found in the Coccidia. In the Hæmosporidia, however, the male and female cells must be removed from the blood of the host to insure further development. This removal is effected by a blood-sucking arthropod, which takes the parasites into its alimentary canal. Here they mature, the male cells forming each a number of microgametes, the female cells each a single macrogamete or egg. The microgametes fertilize the macrogametes, which then metamorphose into elongated elements, the ookinetes. The ookinete pierces the intestinal epithelium of the mosquito and comes to rest in the periintestinal tissue. There is first a period of growth, during which the element assumes a spherical form and becomes inclosed by a delicate wall. This stage corresponds to the oocyst of the Coccidia, but no actual cyst is developed. Eventually the sporozoites are produced directly from this protoplasmic mass, the spore stage being omitted. Excepting for the fact that fertilization is postponed until after the removal of the parasite from the vertebrate host, that part of the acystosporean life history which is passed in the mosquito corresponds exactly to that part of the coccidian life history which is passed in the cyst.

The various species of Hæmosporidia are parasites of the blood of mammals, birds, reptiles, and batrachia. Minchin remarks as follows concerning the Hæmosporidia:

The effects produced * * .* seem to differ markedly in the case of cold-blooded and warm-blooded animals. In the former, there is no evidence that these parasites, however numerous, produce any pathological effect upon their hosts at all, but in birds and mammals they cause fevers and agues of various kinds, of which those that trouble the human species are naturally the best known. At least three types of (malarial) fever are generally recognized, each caused by a distinct form of parasite—the two so-called benign intermittent fevers, tertian and quartan agues, and the pernicious estivo-autumnal fever or tropical malaria. In each case the parasite is introduced into the human body by the bite of a mosquito, and not, so far as is known, in any other way. After a period of incubation, varying from 6 to 12 days, according to the species of parasite, the fever makes its appearance. In the benign forms, the

PLATE XL.—LIFE CYCLE OF THE MALARIA (HUMAN) PARASITE.

1. Free sporozoite, either in salivary glands of the mosquito or in blood of man.
2. Penetration of the sporozoite into a red blood cell.
3 to 6. Growth of trophozoite.
7, 8. Division of trophozoite which brings about destruction of the blood cell and the release of the merozoites in the blood stream. The free merozoites then enter new blood cells, and this cycle may be repeated many times. Finally, however, the sexual cycle is initiated as follows:
9a to 12a. Growth and differentiation of female cell.
9b to 12b. Growth and differentiation of male cell.
13a, 13b. The male and female cells are swallowed by a mosquito.
14a. Maturation of female cell.
14b. Formation of microgametes.
15b. Free microgamete.
16. Fertilization.
17. Ookinete.
18, 19, 20. The ookinete attacks and penetrates a cell of the intestine of the mosquito, and passes completely through the epithelium, coming to rest in the peri-intestinal tissue. (There is not, in life, the reduction in size indicated by the figure.)
21 to 25. Stages in the development of the cyst and formation of the sporozoites.
26. Migration of the sporozoites.
27. Sporozoites in the salivary glands of the mosquito.
13c to 17c. These figures portray the cycle which is supposed to account for cases where malaria is latent, for a longer or shorter period. Ordinarily, unless removed by a mosquito, the differentiated male and female cells (12a and 12b) die, but under certain conditions the latter may continue to live in the blood, to give rise to a renewal of the disease.



LIFE CYCLE OF THE MALARIA (HUMAN) PARASITE. (FROM MENSE'S "HANDBUCH," AFTER GRASSI AND SCHAUDINN.)

feverish symptoms appear at regular intervals, dependent on the time occupied by a complete reproductive cycle of the parasite. Thus in the parasite of tertian ague the schizogony takes 48 hours, and the fever recurs every other day. In quartan ague the schizogony takes 72 hours, and the attacks of fever occur every three days. There may, however, be double or triple infections, the result of distinct inoculations; or again there may be mixed infections of the two forms, so that distinct generations of the parasites occur contemporaneously in a given patient, producing every possible variation in the frequency of the attacks of fever. In pernicious malaria, on the other hand, the sporulation takes place irregularly, and the fever is consequently irregular or continuous in its manifestations. In all cases the fever coincides in its appearance with the actual sporulation of the parasite, when vast numbers of the merozoites are set free in the blood and are attacking fresh, healthy corpuscles. The result of the rapid multiplication of the parasite in the blood, and the consequent destruction of the corpuscles, is a condition of anemia which tends to produce general cachexia, and may terminate fatally. At the same time the melanin granules produced by the parasite and dispersed in the blood when the sheltering corpuscle disintegrates and the merozoites scatter becomes deposited in the spleen and liver, which becomes hypertrophied, and also in the lungs, kidney, and brain, causing a pigmentation of these organs. In pernicious malaria death may ensue from the accumulation of the parasites in the brain to such an extent that the circulation is hindered or completely blocked.

Acystosporea.—In this group of Hæmosporidia, sometimes termed Plasmodidæ, the trophozoite remains in the blood cell which it entered as a sporozoite or merozoite until it divides to form another generation of merozoites, as already described in the account of the typical life cycle of the Hæmosporidia.

The parasites of human malaria are the most prominent members of the Acystosporea and belong to the genera *Plasmodium* and *Laverania*. *Proteosoma* is the form causing malaria in birds, a disease a good deal like that of man. So far as is known, none of the malarialike parasites attack any of the domesticated animals. In addition to the genera just mentioned, Doflein also includes in the Plasmodidæ, *Achromaticus*, a parasite of bats, and *Hæmocystidium*, a parasite of reptiles.

Hæmosporea.—The Hæmosporea, commonly known as hæmogregarines, differ from the Acystosporea principally in that the trophozoite, instead of remaining constantly within the blood cell which it originally attacks, may abandon the cell and live free in the blood plasma, later returning to another cell.

The hæmogregarines include the genera *Lankesterella* and *Karyolysus*, whose species are parasites of batrachia and reptiles, and the genus *Hæmogregarina*, the species of which parasitize all the groups of vertebrates. The carriers for this genus are leeches, ticks, lice, and fleas. It is more or less problematical whether this genus is pathogenic. The genus *Leucocytozoan* is morphologically a good deal like *Hæmogregarina*, but differs in that it selects white blood

cells in which to live. Its members have only recently been brought to attention. They are parasites of mammals, birds, and batrachians. They seem to be quite pathogenic, but so far the work done on them has not been sufficient to determine their economic importance.

Two other genera of hæmosporidian parasites are generally put under the Acystosporea, although their affinities with this group are very doubtful. There is, however, no question as to their economic importance. These genera are *Piroplasma* and *Leishmania*.

PIROPLASMA.

PIROPLASMA BIGEMINUM.

Piroplasma bigeminum (fig. 74) is the agent of Texas fever. This disease occurs in North and South America, Cuba, Porto Rico, South Africa, the Philippines, and Australia. The carrier is the cattle tick (*Margaropus annulatus*), and the mode whereby the transfer is made is almost unique. The cattle tick, which seeks its host as a larva, never afterwards abandons it, but undergoes its metamorphoses and completes that part of its life history upon the animal ordinarily in the exact spot to which it first attached itself. Hence there is no opportunity for a given tick to transmit Texas fever. But by a remarkable adaptation this end is attained. During the last few days spent upon the animal, the female ticks, already fertilized, absorb an enormous amount of blood and become the so-called engorged females. As such they fall to the ground and some days later lay eggs, from which the next brood is hatched. In some unknown manner the parasites taken up with the blood get into the eggs, and hence are already present in the larval ticks when these are hatched. In other words, infection of the tick by the parasite is hereditary, and hereditary infection, in spite of a rather widespread impression to the contrary, is one of the rarest phenomena known.

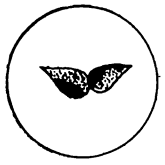


FIG. 74.—*Piroplasma bigeminum*. (After Doflein.)

It results from this mode of conveyance that Texas fever can be controlled much more readily than those diseases of which the carrier visits one host and then another, as, for instance, a fly. It is to be remembered, however, that a pasture in which ticky cattle harboring *Piroplasma* have been is not safe until all the ticks coming from these cattle, as well as the larvæ hatched from such eggs as they have laid, have died. This, in a warm region, may be a number of months.

The carriers of Texas fever in South America and Africa differ both from one another and from the species of the United States, but they are all very closely related, and the life history of each one is much the same.

PIROPLASMA PARVUM.

Piroplasma parvum is the agent of Coast fever or Rhodesian fever. This is a virulent disease of cattle which occurs in Africa, Transcaucasia, and the Philippines. The carriers are the ticks *Rhipicephalus appendiculatus* and *R. simus*. Hereditary infection of these by the parasite has not been demonstrated, and it is not necessary, since these ticks abandon their hosts at the time of the metamorphoses and hence may carry the disease from one cow to another; that is, a tick which passes its larval stage on an infected animal may infect another animal upon which it passes its nymphal stage, and likewise a nymph grown on an infected animal may infect another animal upon which it lives as an adult.

PIROPLASMA OVIS.

Piroplasma ovis causes in sheep a disease known as carceag or ictero-hematuria. It has been described from Hungary, Germany, Roumania, the Balkan Peninsula, Italy, the West Indies, and South Africa. The tick *Rhipicephalus bursa* is the agent of transmission of carceag.

A disease resembling carceag occurs rather rarely among sheep in the western United States, but little is known concerning it or its agent of transmission.

PIROPLASMA EQUI.

Piroplasma equi causes biliary fever in horses. Its range is South Africa, Madagascar, and parts of Europe. The carrier is a tick, *Rhipicephalus evertsi*.

PIROPLASMA CANIS.

Piroplasma canis attacks dogs, the name given to the disease being malignant jaundice. It occurs in Europe, Africa, and India. It is carried by different ticks in different parts of its range; *Hæmophysalis leachi* in Africa, *Rhipicephalus sanguineus* in India, and *Dermacentor reticulatus* in Europe. Canine piroplasmosis probably exists in this country, but as yet satisfactory evidence of its occurrence has not been obtained.

GENERAL REMARKS ON PIROPLASMOSES.

Piroplasmoses are serious diseases, with a high death rate. Apparently all of them, however, may occur in two forms, the chronic and the acute. The former is well illustrated by Texas fever. As a result of the prevalence of the cattle tick in the Southern States nearly all southern cattle harbor the fever parasite in their blood, but do not suffer acutely, since, as already stated, they have undergone a sort of natural vaccination as young calves. But if infected ticks be placed on an adult cow from the Northern States, and the animal thus becomes inoculated with the *Piroplasma*, the chances are that she will die within a short time.

PLATE XLI.—EVOLUTION OF THE PARASITE OF KALA-AZAR.

Figs. 1 to 5. Parasites of kala-azar.

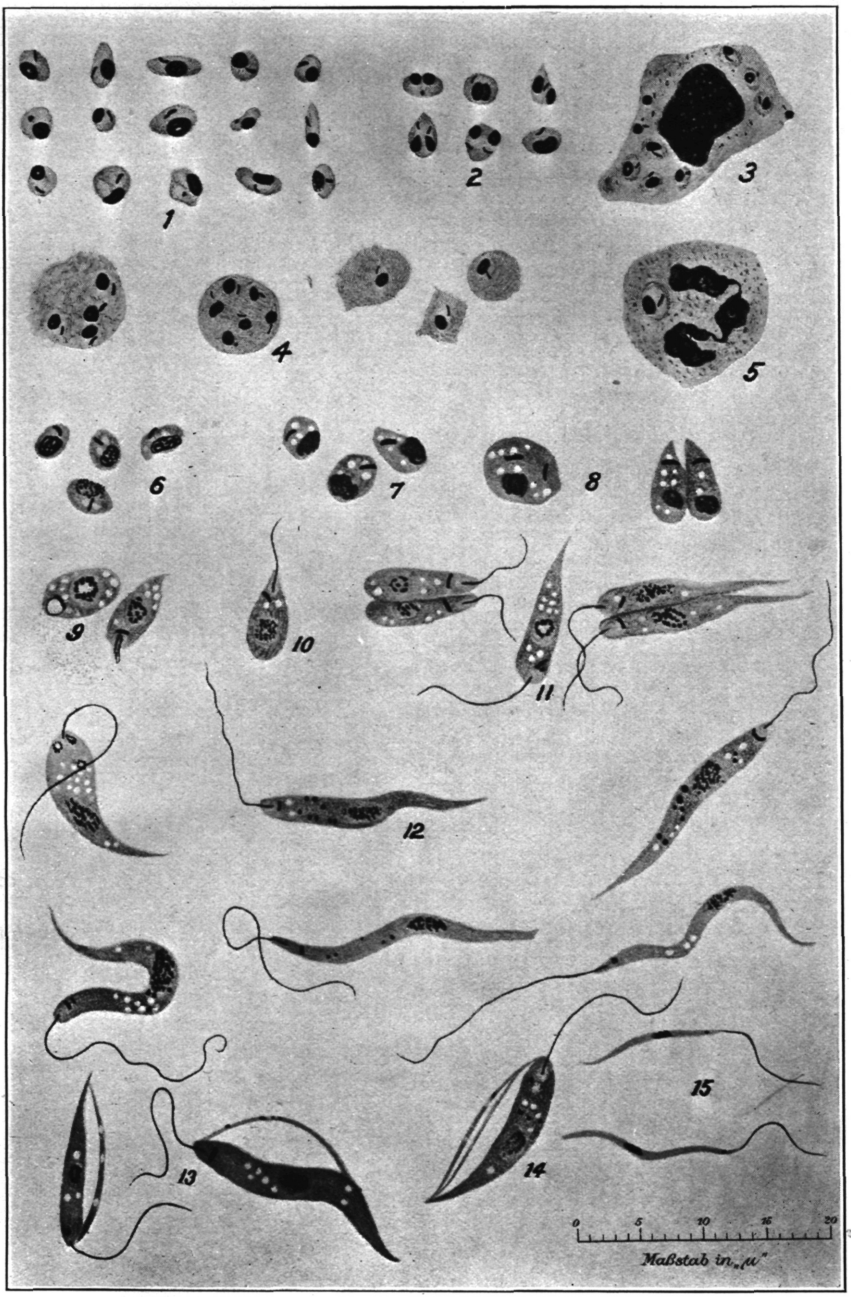
1. Isolated parasites of different forms in the spleen and liver.
2. Division forms from liver and bone marrow.
3. Mononuclear spleen cells containing the parasites.
4. Groups of parasites.
5. Phagocytosis of a parasite by a polynuclear leucocyte.

Figs. 6 to 15. Parasites from cultures.

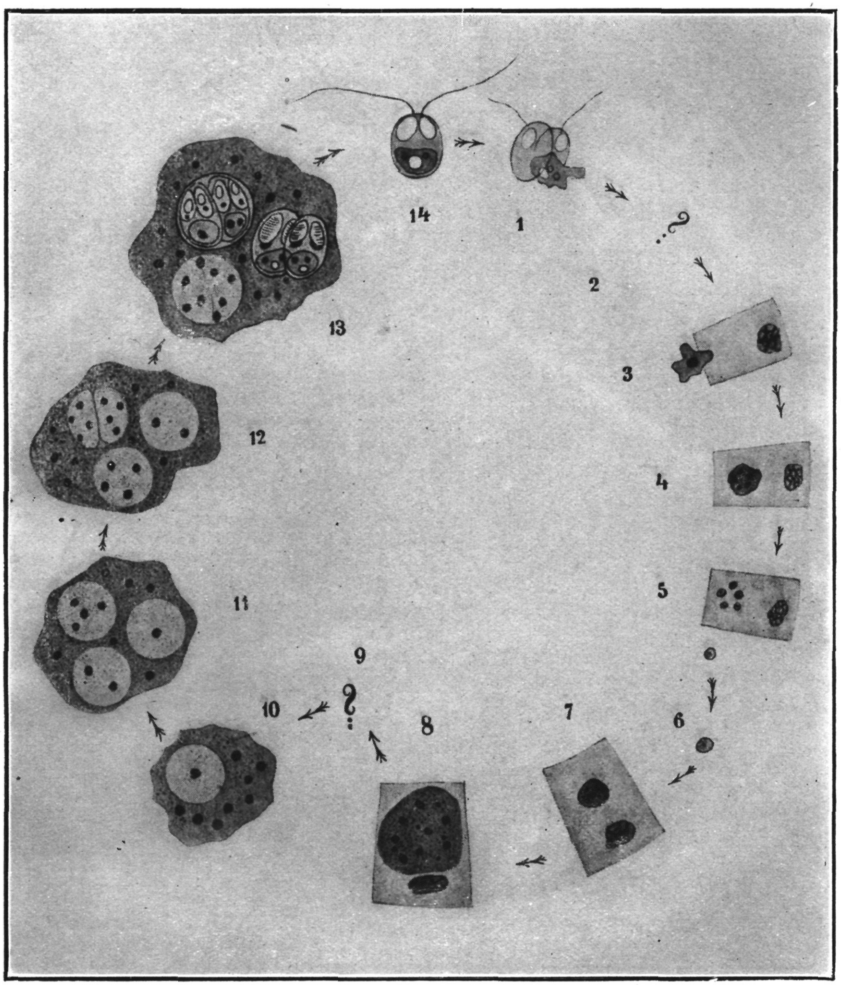
6. First changes in the parasites. The protoplasm has increased in bulk and the nucleus has become larger.
7. Further increase in size. Vacuolization of the protoplasm.
8. Division of the enlarged parasite.
9. Evolution of the flagella.
10. Small piriform parasite showing flagellum.
11. Further development and division of the parasite.
12. Flagellated trypanosome-like form.

13, 14. Flagellated forms dividing by a splitting-off of a portion of the protoplasm.

15. Narrow flagellated parasites which have arisen by the type of division shown in Figs. 13 and 14.



EVOLUTION OF THE PARASITE OF KALA-AZAR. (FROM MENSE'S "HANDBUCH," AFTER LEISHMAN.)



SCHEMATIC DEVELOPMENTAL CYCLE OF A MYXOSPORIDIAN. (FROM DOFLEIN.)

1. Spore opening spontaneously, from which the amebula is creeping. 2. Place where conjugation, if present, may occur. 3. Entrance of the amebula into an ephithelium cell. 4. Nuclear multiplication. 5. Multiple division. Each small body consists of a nucleus with a portion of the protoplasm of the mother cell. 6. Migration of the products of the division. 7, 8. Growth of these in a new cell. 9. Place where conjugation, if present, may occur. 10. Formation of sporoblasts, i. e., the elements from which the spores develop; one such shown. 11, 12. Further development of the sporoblasts; three shown. 13. Further development; two of the sporoblasts have become spores. 14. Ripe spore.

Thus, in a region where piroplasmosis is present, the stock animals native to that region seldom suffer from the acute form of the disease. When, however, such animals are moved to a region where the disease does not exist, the disease tends to break out in the acute form amongst the susceptible animals of that region, causing heavy loss. It is the same when susceptible animals are taken into an infected region; they promptly acquire an acute form of the disease.

It is the last fact which stands in the way of the improvement of cattle in the Southern States. Unless first immunized, northern cattle can not safely be taken into the Southern States, and hence the advantage to be gained by the infusion of fresh blood into southern animals is lost.

LEISHMANIA.

In the genus *Leishmania* belongs the parasite of a tropical disease of man, known as kala-azar. It occurs endemic in many parts of Hindustan and in Burma, China, Arabia, Upper Egypt, Tunis, and Algeria. It has occurred as an epidemic in Assam. It is ordinarily fatal. (See Pl. XLI.)

The parasite of kala-azar is a minute rounded, oval, or pear-shaped organism, which is found generally throughout the body of the patient, but most abundantly in the liver, spleen, and bone marrow. It has a distinct cuticle. The cytoplasm is often vacuolated, and there are two nuclei. These bodies multiply either by simple fission or endogenous multiplication.

The most remarkable feature about these Leishman-Donovan bodies, however, is the fact that if placed in appropriate culture media they evolve into herpetomonads. That is, within the human body they are Sporozoa, much like piroplasms, whereas in culture tubes they become flagellates. Hence the exact zoological position of this parasite is a matter of considerable doubt, some authors placing it in the genus *Herpetomonas*, among the Mastegophora; others calling it *Leishmania* and considering it to be allied to *Piroplasma*.

The disease seems to be carried by the bedbug. Bedbugs fed on kala-azar patients later show in their intestines flagellated bodies resembling those found in the culture tubes.

NEOSPORIDIA.

This group of sporozoa is imperfectly known. Zoologically it is divided into three orders, the Sarcosporidia, Myxosporidia (see Pl. XLII), and Haplosporidia, only the first of which is represented among the parasites of domesticated animals. To Myxosporidia belong a number of forms which cause diseases very destructive to fishes. The parasite causing the disease of silkworms, known as pebrine, which has produced losses in the silkworm industry of France amounting to not less than $200,000,000, and a closely related para-

site which causes malignant dysentery in bees, are both members of the order Myxosporidia. The Haplosporidia appear to be of comparatively little economic importance.

SARCOSPORIDIA.

Sarcosporidia are almost exclusively parasites of the striped muscles of mammals and birds. The trophozoite is elongated and inclosed in the early stages of its development by a delicate cuticle which later becomes an envelope of complicated structure. Spore formation begins at an early stage of development, and is carried on during the growth of the trophozoite. The spores, which are produced in enormous numbers, are very small banana-shaped or spindle-shaped bodies, each containing a nucleus. There may be a delicate cuticle, but, unlike the Coccidia, no protective shell is formed.

Sarcosporidia are common in domesticated mammals and are nearly always present in the pig and the sheep. They are also frequently present in the horse and the ox, and have been recorded from man. In birds they attack chickens and ducks, and have frequently been found in wild birds.

The earliest known stage of the parasite, known as a Miescher's tube, is shown in Plate XXXVII, figure 2. The muscles invaded are more usually those of the trunk in the vicinity of the alimentary canal; first those of the esophagus, then those of the larynx, the body wall, and the diaphragm, and the psoas muscles. In acute cases all skeletal muscles may be affected.

Within the muscle fiber the parasite grows until it distends the fiber to several times its normal breadth, and may finally rupture the fiber sheath and come to lie in the adjacent connective tissue. In this way the invaded muscles are more or less destroyed, necessarily to the injury of the host, and in addition it has been found that Sarcosporidia form a very poisonous toxin, which has been named sarcocystin by Laveran and Mesnil.

The mode whereby Sarcosporidia are transmitted from host to host is not known. It has been found that if mice be fed on the flesh of other mice containing Sarcosporidia they become infected after an incubation period of about six weeks. Since sarcosporidian infection is fatal to mice, and since mice nibble at or even completely devour their dead, this case presents no difficulty; but such a mode of transfer can not well be called into account for infections in cattle, horses, or sheep. The problem is one which requires further study.

The following members of the group may be considered:

SARCOCYSTIS MIESCHERIANA.

This species occurs in swine. The trophozoites may reach a length of 4 millimeters, with a breadth of 3 millimeters. Almost any

muscle may be attacked. The earlier students credited this species with causing a serious although not fatal disease in swine, but it is doubtful if this is so. An extensive invasion of the muscles of the body and hind quarters would, however, render the flesh undesirable for food.

SARCOSCYSTIS BERTRAMI.

This form parasitizes the horse. The parasites reach a length of 9 to 10 millimeters. In heavy infections there is a rather extensive destruction of the muscles, and by some authors this parasite is said to be the cause of a disease of young horses.

In the Western States of America, also, certain ailments of horses have been credited to sarcosporidian infection.

SARCOCYSTIS TENELLA.

This species (see Pl. XXXVII, figs. 3 and 4) is a parasite of the sheep, and may reach a length of one-half inch. In certain regions of Europe nearly all sheep are infected. Its usual situation is the muscles of the throat, but it may also be present in many other parts of the body, among which are the heart muscles. In Europe this form is credited with being quite pathogenic for sheep and frequently a cause of death.

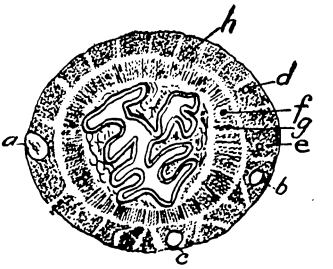


FIG. 75.—*Sarcocystis blanchardi.* Cross section of the esophagus of a cow (natural size). *a, b, c, d,* and *e* are cysts in the outer muscular coat; *f, g,* and *h* are cysts in the inner muscular coat. (After Doflein.)

SARCOCYSTIS BLANCHARDI.

This species was first observed in Java in the zebu, and is not uncommon in domesticated cattle throughout the world. It may occur in nearly any muscle, and it is said to be constantly present in the tongues of Sicilian cattle. (See fig. 75 and Pl. XXXVII, fig. 2.)

Sarcocystis lindemanni is the form which has occasionally been found in man.

In mice Sarcosporidia are by no means rare, and the infections are apparently always fatal. Heavily infected mice are sluggish and present a fat, puffy appearance, due to the distension of the muscles by the parasites. This is seen in Plate XXXVII, figure 5, which shows the extent to which the parasite may be present, nearly every muscle bundle being invaded, and it is indeed remarkable that muscles so completely invaded are able to perform their usual functions.

Sarcosporidia have never been taken much into account in considering the diseases of domesticated animals, since they never cause epizootics and are apparently never fatal. In the last few years, however, it is beginning to be suspected tha httey are quite dangerous parasites. Thus, very recently the camels of northern Africa were found to be extensively parasitized and to be thereby greatly debilitated. The group is deserving of much more attention than it has hitherto received.

CILIATA.

This group of Protozoa is composed of a large number of species, nearly all of which are free-living. Ciliates are universally distributed, occurring wherever there is water. When water containing organic matter is exposed to the air for a few days it will be found to be swarming with ciliates.

Scarcely any of the ciliates are parasitic, and it is questionable whether any are harmful parasites, with the possible exception of certain forms which are ectoparasitic on fishes.

In the paunch of ruminants are found numerous species of ciliates, and these are almost constantly present in very great numbers. They appear to do no damage, and indeed the possibility is not excluded that their presence may be actually beneficial to their hosts through some favorable influence which they may have on the digestive processes.

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